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Life vs Limb: The Compartment Syndrome Conflict

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Case Presentation

- A 24-year-old, male patient, status post MVC rollover in Mexico, was transferred to San Antonio Military Medical Center by air. His major injuries comprised of right tibia and fibula fractures, multiple rib fractures, and liver laceration. Diarrhea and vomitus positive for blood, and was transfused 2 units of PRBCs en-route. He was also noted to have elevated potassium of 7.5 mEq/L.
- His only known past medical history consisted of alcoholic cirrhosis, otherwise unknown further medical, surgical, allergy history, NPO status.
- Upon arrival he was noted to be GCS 15, obese, agitated, diaphoretic and pale, with vitals showing hypotension and tachycardia
- Ultrasound revealed a positive FAST exam and a cirrhotic liver.
- Abdomen was tender to palpation throughout and right lower leg was noted to be extremely swollen with indeterminate pulses.
- The trauma surgery team wanted to proceed with both emergent ex-lap and subsequent fasciotomies given the clinical diagnosis of compartment syndrome.
- Given hemodynamic instability, he was emergently intubated in the trauma bay and transported to the OR for exploratory laparotomy and fasciotomy.

Preoperative and Intraoperative Considerations

- Presumed full stomach - RSI with rocuronium to avoid succinylcholine induced hyperkalemic response
- Class 3 Shock by ATLS Designation –large bore IVs, arterial line, SASAM
- Elevated potassium, likely from prolonged extrication and transport from MVC in Mexico, possibly secondary to lactic acidosis from exsanguination and tissue ischemia or K+ sparing diuretics for treatment of alcoholic cirrhosis. Avoidance of K+ sparring diuretics and maintenance of MAP to avoid further ischemia
- Multiple lower long bone fractures with tense lower extremity compartments
- Unknown allergies, medications, past medical and surgical history

Signs and symptoms of Hyperkalemia

- Clinical manifestations usually when > or = to 7.0 mEq/L
- Muscle weakness or paralysis - Ascending muscle weakness that begins with legs and progresses to trunk and arms[1] mimicking Guillain –Barre syndrome[2]and rare respiratory muscle weakness[3]
- Cardiac manifestations - EKG changes – (Tall peaked T waves with shortened QT, progressing to PR and QRS lengthening, concluding with ventricular standstill)
- Reduced urinary acid excretion - Interferes with renal ammonium (NH_4^+) excretion and further contributes to metabolic acidosis seen in trauma patients

Intraoperative Management of Hyperkalemia

- Stabilize the membrane – Calcium chloride (500-1000mg over 3 mins) 3x elemental calcium than calcium gluconate (however can cause local irritation) Repeated every 30-60 minutes because results are shortlived[4]
- Remove excess potassium from body – loop/thiazide diuretics vs. dialysis
  - Up to 40mg of IV furosemide in diuretic naive patients
  - Avoid in patients with severe renal failure (consider dialysis or CRRT in these patients)
- Drive potassium into the cell
  - IV insulin - Enhances activity of Na-K-ATPase pump function in skeletal muscle
    - Give insulin alone if serum glucose is >250 mg/dL
    - 10 units insulin with 500 mL of 10% dextrose IV
  - Beta-2 agonist – enhances Na-K-ATPase pump and has additive effect with insulin
    - Expected K drop 0.5 to 1.5 mEq/L (1.2 to 1.5mEq/L with insulin)
    - Given in 4-8x dose used for bronchodilation (8-16 puffs)[5]
  - Hyperventilation - shifts H^+ out of cells, driving K^+ into cells
- Treat reversible causes of hyperkalemia
  - ACEi, ARBs, chronic heparin therapy, NSAIDs inhibit aldosterone release and reduce urinary potassium excretion
  - Metabolic acidosis, beta blockers, and insulin deficiency cause increased potassium release from cells

Compartment Syndrome Implications

- Risk Factors – long bone fractures, most commonly tibia (75% of cases)
- Delay in fracture resolution or closed reduction can increase pressures
- Blood flow cannot meet demands of surrounding tissue, venous outflow is reduced, tissue edema ensues with further rise in pressure
- Signs: pain, pallor, paresthesia, paralysis, with late sign-pulselessness
- Gold standard for diagnosis is compartment pressure
- The definitive treatment is emergent fasciotomy (compartment release)
- Release causes washout of electrolytes (namely K^+), myoglobin, inflammatory cytokines, and can result in rhabdomyolysis and DIC

Case Management

- Despite optimal medical management (hyperventilation, albuterol, furosemide, insulin with glucose, calcium) patient's potassium ranged between 6.8 and 7.0.
- His abdomen was packed and his right lower leg noted to be extremely swollen with indeterminate compartment pressures and marginally Doppler-able pulses
- A discussion between the lead trauma surgeon and anesthesia team regarding the risks of compartment release while patient remained hyperkalemic despite maximal management, resulted in halt of further surgery and transport to ICU for emergent dialysis for return to OR once potassium normalized.
- This conflict, exemplifies the need for good communication between anesthesiologist and surgeons and ultimately led to not only sparring of the limb, but potentially saving the patient's life.

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